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ABSTRACT

Traditional medical interventions for the management of pain have consisted largely of either pharmacological treatments or surgery to interrupt the involved neural pathways. The results of these procedures have been largely unsatisfactory because of debilitating side effects and recurrence of pain. Investigations of a host of psychological factors related to the pain experience, however, have spawned a variety of cognitive-behavioral treatments which have shown considerable promise in assisting people in coping with pain. This paper discusses the neurophysiological mechanisms which underly the pain phenomenon and which may account for some of these promising treatment effects. (Author)

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THE COGNITIVE-BEHAVIORAL MANAGEMENT OF PAIN:

Neurophysiological Relevancies

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THE COGNITIVE-BEHAVIORAL MANAGEMENT OF PAIN:

NEUROPHYSIOLOGICAL RELEVANCIES

Pain is clearly one of the most universally recognized and important concerns of mankind. While acute pain often serves the critically useful function of warning the person of incipient disease or trauma, severe pain of the chronic variety exacts an incalculable toll of physical suffering and emotional anguish, vocational displacement and economic loss on those so afflicted. In spite of this widespread recognition, many aspects of the phenomenon of pain remain enigmatic to clinicians and researchers alike. Bonica (1973) chides practitioners for their traditionally narrow view in attempting to understand pain and ameliorate its effects. Traditional medical interventions for the management of pain have consisted largely of either pharmacological therapies or surgical insult to the involved neurological tracts. The results of these procedures have been far from satisfactory because of such debilitating side effects as narcotics addiction, tissue mutilation and loss of somatic function, and because of a disheartening tendency for pain to recur and return to pretreatment levels.

The last 20 years, however, have witnessed an evolution of research documenting the existence of a host of psychological factors related to pain. Such investigations have spawned a variety of methods in the cognitive-behavioral literature which have been touted as potentially useful packages for assisting people in coping with inescapable noxious discomforts ranging from routine tooth prophylaxis to childbirth. A clue as to the course of this evolution is provided by Murray (1971)

in his preference for the term "pain experience," which integrates all the effects of noxious stimulation: (a) reactions to the threat of pain; (b) reactions to the stimuli present; (c) the pain sensation as well as accompanying sensations -- e.g. heat, cold, pressure; and (d) reactions to these sensations. The psychological factors influencing these effects--including interpretations of the physical sensations, past experience, and anxiety -- strongly influence the individual's perception of, and responses to, the pain experience.

Using the phrase "psychophysiology of pain," Sternbach (1975) urges that the treatment and rehabilitation of pain patients should revolve around three disciplines -- psychology, sociology, and physiology -- each contributing its own concepts and methodologies. Several critical reviews of the growing liturgy of such comprehensive strategies highlight both their enormous future clinical potential, and their present fledgling state of development (cf., Meichenbaum & Turk, 1976; Melzack, 1973; Weisenberg, 1977; Weisenberg & Tursky, 1976).

While there has been some clear progress through this state of development, a number of definitional, methodological, and interpretational difficulties exist. The pain experience is a highly individualized one, and a unified definition of pain is bound to be simplistic at best. While some researchers have restricted the meaning of the phenomenon of pain to a unique sensation along with other sense modalities (e.g. Mountcastle, 1974) others have viewed it as an emotional or motivational state which demands attention (see Murray, 1971). The distinctions are not purely semantic, for as Weisenberg (1977) reminds us, definitions of pain "must deal with both aspects of pain, especially as

it (pain) relates to pain of clinical, pathological origin." (p. 1009)

Numerous methodological difficulties have obscured the evaluation and comparison of pain management techniques. Even in controlled laboratory settings, general perspectives from different studies are hard to sketch. Pain is induced in laboratory subjects in different ways. The proverbial college freshperson has: had hands, feet and genitalia immersed in ice water (Farr, 1974; Horan & Dellinger, 1974; Horan, Hackett, Buchanan, Stone, and Demchik-Stone, 1977; Stone, Demchik-Stone, & Horan, 1977); been blasted with loud noise (Epstein & Clark, 1970); been subjected to electrocution (Blitz & Dinnerstein, 1968; Buss & Portnoy, 1967; Craig & Weiss, 1972; Staub & Kellett, 1972); had his/her finger smashed with plexiglass wedges (Barber & Cooper, 1972) or zapped with ultrasound (Gelfand, 1964); had a wooden dowel slowly pressed into a shin (Farr, 1974; Horan, Hackett, Buchanan, Stone & Demchik-Stone, 1977); had circulation to a limb impeded by tourniquet (Smith, Lowenstein, Hubbard & Beecher, 1968); been burned (Hardy, Wolff & Godell, 1952); had a balloon inflated in his/her esophagus (Chapman & Jones, 1944); and has had chemical irritants injected under the skin (Lim, 1968). An accounting of our hapless frosh's agonies has been projected onto innumerable self-report scales, stopwatches, ammeters, pressure guages, and physiological measuring devices. This inquisition has produced a veritable army of sore subjects and a vast sea of data for a score of avid researchers. The interpretational difficulties in sifting through the literature are considerable. Moreover, experimental sessions themselves are often saturated wtih factors that confound

treatment effects: experimenter sex, types of instructions, subjects' expectations, the presence of other subjects, and the kinds of pain coping skills that subjects bring into the experimental cubicle from the outside world. In relatively uncontrolled clinical settings, where laboratory conditions and accepted experimental procedures are often unfeasible and/or unethical, one could only expect these problems to be magnified.

Further ambiguities are encountered when one attempts to understand pain and its management from the physiological viewpoint. Not surprissingly, neurologists, neurophysiologists and neuroanatomists are not in total agreement in their description of the pain experience. However, some relatively new findings in these disciplines may have important implications for cognitive-behavioral strategists and their work. This paper reviews, naturally rather superficially, the evolution of the modern theories of pain reception and transmission, and integrates these theoretical concepts with the findings of pain management research.

Melzack (1973) describes the evolutionary history of pain theories under three reasonably discrete classifications: affect theories, specificity theories, and pattern theories. While the first of these had its roots in the Aristotelian era, clearly predating its competitors, it seems to have fallen out of grace with pain researchers, perhaps because of its radical tenets. The proponents of the affect theories considered pain to be an emotion--the opposite of pleasure--rather than a sensation, and all sensory inputs could contain a dimension of pain. While some extreme perspectives were pushed off the

field, Melzack argues that this view does contain important (though heretofore neglected) ramifications both for those who study pain, and for those who suffer from it. Those who distill from the broad rubric of pain such components as the "reaction to pain" (Beecher, 1959; Murray, 1971) give great credence to its affective dimensions. Thus, pain is not simply a perceptive event; it also has a strong negative affective quality which drives us into action to stop it.

The most traditional view of pain perception is known as "specificity theory." Specificity theory, the most classical description of which was probably provided by Descartes more than three centuries ago, is still prevalent throughout much of the medical literature in spite of more recent and powerful theories (Melzack, 1973). Specificity theories propose that the free nerve endings which pervade the skin are specific "pain receptors" while the more complex receptor organs subserve the other sense modalities: touch, heat, and cold. Each cutaneous pain receptor is assumed to have a sensitive area on the skin above it, and to project to a discrete pain center in the brain. While a host of clinical factors defy explanation in terms of a rigid specificity model, it is clear that these early theorists made important contributions that should be retained in further theoretical formulations of pain.

The pattern theories, again according to Melzack (1973), evolved around the turn of the century seemingly as a reaction against the unverifiable assumptions implicit in specificity theories. The simplest version, peripheral pattern theory, posits that excessive peripheral stimulation generates particular patterns of nerve impulses

which evoke pain by summation of the skin sensory input at the dorsal horn cells in the spinal cord. Later histological evidence demonstrating the high degree of pain receptor fiber specialization led to a more tenable version of this conceptual model known as central summation theory, in which summation of pain producing stimuli occurs at the level of the brain itself, probably via synchronized firing in thalamocortical neural circuits. The primary evidence for this is the frequent failure of surgical spinothalamic cordotomy (even bilateral cordotomy) in abolishing many chronic pain states (e.g., phantom limb pain).

Melzack (1973), after a comprehensive analysis of existing theories of pain perception and transmission, suggests refinements for further theorizing.

> Any new theory of pain, it is now apparent, must be able to account for:
>
> 1. The high degree of physiological specialization of receptor fibre units and of pathways in the central nervous system.
>
> 2. The role of temporal and spatial patterning in the transmission of information in the nervous system.
>
> 3. The influence of psychological processes on pain perception and response.
>
> 4. The clinical phenomena of spatial and temporal summation, spread of pain, and persistence of pain after healing.
>
> (p.153)

The gate-control theory of Melzack and Wall (1965) has attempted to weave these criteria and the contributions of previous theories into a comprehensive mosaic for facilitating a deeper understanding of pain states. Briefly, the gate-control theory posits that descending

influences from the brain modulate the "gatekeeping" function of hypothesized neural mechanisms in the dorsal horns of the spinal cord. These mechanisms, in turn, control the flow of ascending peripheral nerve impulses to the central nervous system. "When the amount of information that passes through the gate exceeds a critical level, it activates the neural areas responsible for pain experience and response" (Melzack, 1973, p. 153).

The exact mechanism which modulates the gating function is, as yet, undemonstrated, but Melzack and Wall (1965) suggest that the relative activity in large-diameter (A-beta) and small-diameter (A-delta and C) neurons, and descending activity from the brain, combine to fine tune the gate and regulate the flow of impulses through it. Impulses transmitted through large fibers are inhibitory in nature, closing the gate and impeding the progression of stimuli to higher centers. Once a critical level of stimulation is reached, small-fiber activity opens the gate and facilitates synaptic transmission of pain stimuli to centrally projecting T (transmission) cells. Melzack and Wall (1970), in discussing more distal afferent routes emphasize the different components of the pain experience and the different neuroanatomical bases of those components. Thus, fibers projecting to the ascending reticular formation and limbic system arouse the aversive cognitive-motivational component of pain which may initiate a flight response. Circuits reaching the somatosensory cortex and ventrobasal thalamus may be responsible for the sensory-discriminative component.

Descending influences from higher cortical centers to the gating

mechanism would appear to be conceptually necessary to account for the influences of such cognitive processes as attention, interpretation, anxiety, experience, and presumably, learned coping mechanisms designed to influence these processes. Melzack and Wall (1965) posit the concept of a central control trigger to regulate these descending influences. While precise anatomical sites for such a mechanism have not been clearly demonstrated, Melzack and Wall (1965) argues that the medial-lemniscal and dorso-lateral systems of the dorsal column are strong candidates. Their evolutionary sophistication, speed of conduction, stimulus precision, and adaptability to stimulus changes make these structures uniquely suited to the task.

Clearly the gate control theory is not without its critics (see Weisenberg, 1977), and will undoubtedly undergo considerable revision as the incoming data warrant. It does, however, possess a conceptual and explanatory elegance which accounts for much of the puzzling aspects of clinical pain states. It has stimulated new interest in pain research and management, and has provided us with a tentative neurological vehicle for exerting psychological influence over noxious stimulation.

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